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# Development of a fluid-structure interaction model to simulate mitral valve malcoaptation

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## Abstract

Mitral regurgitation (MR) is a condition in which the mitral valve does not prevent the reversal of blood flow from the left ventricle in to the left atrium. This study aimed at numerically developing a model to mimic MR and poor leaflet coaptation and also comparing the performance of a normal mitral valve to that of the MR conditions at different gap junctions of 1, 3, and 5 mm between the anterior and posterior leaflets. The results revealed no blood-flow to the left ventricle when a gap between the leaflets was 0 mm. However, MR increased this blood-flow with increases in the velocity and pressure within the atrium. The pressure within the aorta did not vary meaningfully though (ranging from 22 kPa for a 'healthy' model to 25 kPa for severe MRD). The findings from this study have implications not only for understanding the changes in pressure and velocity as a result of the MR in the ventricle, atrium, or aorta, but also for the development of a computational model suitable for clinical translation when diagnosing and determining treatment for MR.



**Keywords:** blood flow; fluid-structure interaction; left atrium; left ventricle; Mitral regurgitation.

# Introduction

Mitral regurgitation (MR) is a condition in which the leaflets of the mitral valve do not prevent backflow of the blood from the left ventricle to the left atrium **[1],[2],[3]**. This is often due to poor closure, or coaptation, between its leaflets; known as mal-coaptation. Thus, if MR is severe, the pressure of the left atrium increases leading to symptoms such as dyspnea, fatigue, orthopnea, and pulmonary edema **[4]**. Although annular dilation, and its subsequent effect on coaptation, has been assessed *in vitro* **[5]**, there is limited understanding of the effect of the specific mode of mal-coaptation on the subsequent hemodynamic characteristics of the blood flow during MR between the left atrium, left ventricle, and the aorta. MR can be studied clinically, but it does not offer the possibility to assess variables associated with mal-coaptation independently to assess how they contribute to subsequent hemodynamics during MR. Computational modelling, and in particular, transient Fluid-Structure Interaction (FSI) **[6]** offers the potential to determine the mechanisms linking altered mechanics and coaptation between mitral valve leaflets, with the resulting pathophysiological hemodynamics.

There is an extensive range of computational modelling of the mitral valve within the literature. For instance, Wenk *et al*. **[7]** proposed a finite element (FE) model for the left ventricle with MR using magnetic resonance imaging (MRI) data from sheep. Stevanella *et al*. **[8]** presented a patient-specific structural FE model of the mitral valve to assess mitral annuloplasty procedures. A three-dimensional FE model of the mitral valve has also proposed to assess the nonlinear mechanical performance of the anterior and posterior leaflets during the diastole and systole **[9]**. Surgical repair of the mitral valve has been assessed too **[10],** along with the effect of collagen concentration within the mitral valve leaflets **[11]**. More recently, there has been a focus on combining Computational Fluid Dynamics (CFD) with end-diastolic geometry and time-dependent deformation of the left ventricle cavity **[12]**, as well as MR flow **[13]**. CFD has also been used to assess insufficiency through the mitral valve Sonntag *et al*. **[14]**. However, regardless of the depth of literature, currently no such studies have determined a mechanical pathway which links the specific pathophysiological hemodynamic characteristics during MR to the altered leaflet mechanics which result in mal-coaptation. Even an initial step of assessing the influence of a gap between the two mitral leaflets has not been assessed; despite the clinical importance of such studies, and their potential to be used to propose novel repair strategies, both the computational and experimental literature is very much limited **[15]**.

This study was aimed at developing a transient FSI model of the mitral valve able to predict mal-coaptation. The model geometry is based on subject-specific MRI data. For this initial development, a two-dimensional (2D) model has been used. While three-dimensional models are of value [16], the timescales associated with solving 2D models at present more closely match clinical timeframes [17]. This initial model has focused on hemodynamic assessment of blood flow through the left atrium, left ventricle, and the aorta, during mal-coaptation of the anterior and posterior mitral valve leaflets due to gap sizes of 1, 3, and 5 mm.

## Materials and methods

An MRI scan (Siemens, Germany,1.5 Tesla) of a mitral valve was obtained from a 60-year-old male. Ethical approval was obtained from Tehran Modarres Hospital. The mitral valve scanned had normal coaptation. From this scan, distinct MR cases were simulated using a 2D model (**Fig. 1**).

Four cases were simulated. The first model included tightly coapting anterior and posterior leaflets so that no MR occurred across the mitral valve (Case A). The second model included a 1 mm gap between the leaflets, and was considered as a mild MR model (Case B). The third model included a 3 mm gap between the leaflets, representing fully developed MR (Case C). The last model included a 5 mm gap, simulating severe MR (Case D). Left atrial and ventricular systolic pressures were applied as boundary conditions **[18],[19]** where the blood velocity (flow rate if considered over the cross-sectional area of the aorta) was applied as a further boundary condition **[20]**. As the mitral valve is closed during systole, the leaflets were assumed to be fixed in position. This boundary condition would mimic the mitral valve leaflets following the initial elongation of chordae tendineae; consistent with previous studies **[21]**.

Blood flow was assumed to be laminar, incompressible, and Newtonian; a suitable approximation under large-scale flow as occurs within the heart **[22]**. The viscosity of blood was taken to be 5.5 mPa/s with density of 1056 kg/m$^3$ **[23], [24]**. Anterior and posterior mitral leaflets were assumed to behave as linear elastic materials (with a Young's modulus of 1 and 2 MPa, respectively). The density and Poisson's ratio of the leaflets applied to the model were 1060 kg/m$^3$ and 0.488, respectively **[25], [26]**.

Triangular elements were used to generate the mesh. The mesh density analyses have been conducted to select the appropriate mesh size (stress was assessed with mesh density). The number of elements used in the model was 3309. The simulation time was set to run for 0.45 s, i.e. only systole was modelled **[27]**.

FSI was used to perform transient, and simultaneous simulations of the mitral valve. Simulations were performed using Comsol Multiphysics (Comsol Multiphysics, Stockholm, Sweden). The formulation employed led to the following physical restraints/coupling to enable interaction between simulation of fluid flow and structural deformation: a) the deformation of the contact points of the fluid (blood) and solid (Leaflet) were coupled (i.e. rate of change of solid boundary displacement is matched by the velocity of blood); b) the planar force of the fluid in the contact point of the fluid and solid acts as the planar force of the solid (i.e. the loading condition applied to the leaflets); and c) a no slip condition is applied to the fluid boundary **[28]**.

## Results

Velocity contours at 0.2 s (during systole) are provided **Fig. 2** for all four cases. For the 'healthy' model (Case A), MR was not possible **[29]** and a vortex ensued behind the leaflets. When MR was simulated (Cases B-D), the blood volume and the velocity increased due to an increasing gap between the leaflets. The result is blood-flow into the atrium (**Fig. 2**). For mild MR (Case B), the leakage rate was low (around 2.5 m/s). However, blood-flow velocity reached 5 m/s for Cases C and D. For severe MR (Case D), the velocity was such that a large vortex ensued within the left ventricular cavity.

The velocity of blood within the left atrium is provided in **Fig. 3**. For Case A, this velocity was zero. For MR, blood flow increased to peak values of 0.81, 1.63, and 2.42 m/s for Cases B, C, and D, respectively. This trend continued from the time at which these peaks occurred up to the end of the simulation.

The time-dependency of left atrial blood pressure is provided in **Fig. 4**. For Case A, these pressures were in agreement with experimental data **[30]**. For Case B, similar trends were predicted for time-dependent pressures, but a small alteration in the maximum (0.3 Kpa) and minimum (1.9 Kpa) pressures did occur. However, for Cases C and D, an abnormal pressure trend was predicted. This abnormal pressure was related to the rate of systolic ventriculo-atrial blood-flow; in essence, the velocity of MR. Peak pressures for Cases C and D were 2.12 and 2.90 kPa, respectively.

Time-dependent ventricular pressure adjacent to the mitral valve is provided in **Fig. 5**. There is an increased pressure during early systole (to 5 kPa) for both cases C and D. For Case D this pressure peaked at 7.80 kPa. The graphs predicted that the increased size of the gap, C and D cases, led to a ventricular pressure increase of 5 to 7 kPa comparing to normal situation, Case A.

This model has made predictions of altered MR hemodynamics and a potential relationship to left atrial pressure – this might have implications for LV and LA remodeling. Also, there may be some comparisons to literature (e.g. clinical / experimental).

Time-dependent aortic pressure is displayed in **Fig 6**. No meaningful differences in pressure were predicted for aortic blood-flow. These values ranged from peak pressures 22 kPa for a 'healthy' model (Case A) to 25 kPa for severe MR (Case D. Fig 7 represents the pressure contours at 0.2 s (during systole) for all cases.

## Discussions

We have presented a model which has made predictions of altered MR hemodynamics and a potential relationship to left atrial pressure. This might have implications for left ventricle and left atrium remodeling. Comparison of four MR models showed different hemodynamic characteristics based on the size of the gap between leaflets. The blood jet generated small vortexes in mild and severe MRs.

Looking to the pressure contours, we found out that the absence of mitral regurgitation in model-A leads to an increase of systolic pressure in the left ventricle. The results is in agreement with Lassila et al [31] findings. On the other hand, Thomas et al [32] showed that peak ejection velocity was seen to increase due to MR severity. This is also in agreement with our findings. During the ventricular systolic phase the left ventricle contradiction, the rate of blood flow from the mitral is very low or zero [33]. The leaflet close mechanism depends on the distance of ring vortexes of each ventricle during systole [34]. These vortexes could not be modeled in the commercial FE software. Therefore, the leaflets were considered as fixed components. This assumption allowed the mitral valve was closed during systole and the blood flow showed no effect on the leaflets. The limitation of this study was that we considered a fixed left ventricle wall which could not mimic the ventricle failing to reproduce the necessary pressures during severe MR.

We believe that this is the first model which assesses mal-coaptation and its relationship with MR: increasing MR velocity, left atrium pressure and other hemodynamic parameters. We also hypothesize that the reduced aortic flow may not be due directly to MR but to increased difficulty of the heart to pump blood. Although the obtained results were clinically shown but our work is the first to link actual changes in coaptation to specific hemodynamic factors including pressure and velocity. The success of this study opened the way for further development in to a clinical tool. On the other hand, there were several limitations in our study which should be considered for further studies. The inclusion of myocardium as well as endocardium and the influence of these structures should be investigated therefore another fluid structure interaction model is required to show these structures responses and the flow inside the left ventricle. Dynamic motion of leaflets should also evaluated. It is also suggested to make more accurate anatomically model using three dimensional echocardiography or new MRI sets.

## Conclusions

An FSI model of the left side of the heart has been produced, from an MRI scan to study mitral valve mal-coaptation. Increased mal-coaptation resulted in increased left atrial pressure, which would presumably lead to enlargement (as seen clinically). A bigger gap between the leaflets led to a large vortex within the left ventricle and higher blood velocity in the atrium. The pressure adjacent to the mitral valve also increased with increased mal-coaptation. These findings have implications not only for understanding alterations in pressure and velocity as a result of the MR in the ventricle, atrium, or aorta, but also for providing a comprehensive numerical model suitable for clinical translation. We believe that this model, along with improved version, is a powerful tool for future clinical scenarios as it could capture the presence of MR. However, the model should be improved in number of ways as mentioned before.

## Compliance with Ethical Standards

**Funding:** None.

**Conflict of Interest:** None.

**Ethical approval:** Ethical approval was provided under the permission of Modarres Hospital, Tehran, Iran.

## References

[1] Smith PK, Puskas JD, Ascheim DD, Voisine P, Gelijns AC, Moskowitz AJ, Hung JW, Parides MK, Ailawadi G, Perrault LP.Surgical treatment of moderate ischemic mitral regurgitation. N Engl J Med 2014;371(23) : 2178-2188.

[2] Glower DD, Kar S, Trento A, Lim DS, Bajwa T, Quesada R, Whitlow PL, Rinaldi MJ, Grayburn P, Mack MJ. Percutaneous mitral valve repair for mitral regurgitation in high-risk patients: results of the EVEREST II study. J Am Coll Cardiol 2014; 64(2) : 172-181.

[3] Al-Atabi M, Espino DM, Hukins DW. Computer and experimental modelling of blood flow through the mitral valve of the heart. JBSE 2010; 5(1):78-84.

[4] Anwar AM, Folkert J, Soliman OI. Clinical Recognition of Tricuspid Valve Disease.Practical Manual of Tricuspid Valve Diseases, Springer ;2018.Chapter 3.

[5] Espino DM, Shepherd DE, Buchan KG. Effect of mitral valve geometry on valve competence. Heart vessels 2007; 22(2): 109-115.

[6] Espino DM, Shepherd DE, Hukins DW. Transient large strain contact modelling: A comparison of contact techniques for simultaneous fluid–structure interaction. Eur. J. Mech. B/Fluids 2015; 51: 54-60.

[7] J.F. Wenk, Z. Zhang, G. Cheng, D. Malhotra, G. Acevedo-Bolton, M. Burger, T. Suzuki, D.A. Saloner, A.W. Wallace, J.M. Guccione, M.B. Ratcliffe, First Finite Element Model of the Left Ventricle With Mitral Valve: Insights Into Ischemic Mitral Regurgitation, The Annals of Thoracic Surgery 89(5) (2010) 1546-1553.

[8] Stevanella M, Maffessanti F, Conti CA, Votta E, Arnoldi A, Lombardi M, Parodi O, Caiani EG, Redaelli A. Mitral Valve Patient-Specific Finite Element Modeling from Cardiac MRI: Application to an Annuloplasty Procedure. Cardiovasc Eng Technol 2011; 2(2): 66-76.

[9] Prot V, Haaverstad R, Skallerud B. Finite element analysis of the mitral apparatus: annulus shape effect and chordal force distribution. Biomech Model Mechanobiol 2009; 8(1): 43-55.

[10] Votta E, Maisano F, Soncini M, Redaelli A, Montevecchi FM, Alfieri O. 3-D computational analysis of the stress distribution on the leaflets after edge-to-edge repair of mitral regurgitation. J Heart Valve Dis 2002;11(6): 810-822.

[11] Kunzelman KS, Quick DW, Cochran RP. Altered collagen concentration in mitral valve leaflets: biochemical and finite element analysis. Ann Thorac Surg 1998; 66(6):198-205.

[12] Lassila T, Malossi C, Stevanella M, Votta E, Redaelli A, Deparis S. Simulation of left ventricle fluid dynamics with mitral regurgitation from magnetic resonance images with fictitious elastic structure regularization, arXiv:1707.03998 [physics.med-ph] ;2017.

[13] Wang Y, Quaini A, Čanić S, Vukicevic M, Little SH. 3D experimental and computational analysis of eccentric mitral regurgitant jets in a mock imaging heart chamber. Cardiovasc Eng Technol 2017;8(4):419-438.

[14] Sonntag SJ, Li W, Becker M, Kaestner W, Büsen MR, Marx N, Merhof D, Steinseifer U. Combined computational and experimental approach to improve the assessment of mitral regurgitation by echocardiography. J Bioeng 2014;42(5): 971-985.

[15] Al-Atabi M, Espino DM, Hukins DW, Buchan KG. Biomechanical assessment of surgical repair of the mitral valve. Proc Inst Mech Eng H 2012; 226(4): 275-287.

[16] Bahraseman HG, Hassani K, Khosravi A, Navidbakhsh M, Espino DM, Kazemi-Saleh D, Fatouraee N. Estimation of maximum intraventricular pressure: a three-dimensional fluid–structure interaction model. Biomed Eng Online 2013;12( 122).

[17] Bahraseman HG, Languri EM, Yahyapourjalaly N, Espino DM .Fluid-structure interaction modeling of aortic valve stenosis at different heart rates. Acta Bioeng Biomech 2016;18 (3):11-20.

[18] Burriesci G, Howard I, Patterson E. Influence of anisotropy on the mechanical behaviour of bioprosthetic heart valves. J Med Eng Technol 1999; 23(6): 203-215.

[19] Burriesci G, Marincola FC, Zervides C. Design of a novel polymeric heart valve. J Med Eng Technol 2010; 34(1): 7-22.

[20] Hall JE. Guyton and Hall textbook of medical physiology. Philadelphia: Saunders Elisever; 2011.

[21] Espino DM, Shepherd DE, Hukins DW. Evaluation of a transient, simultaneous, arbitrary Lagrange–Euler based multi-physics method for simulating the mitral heart valve. Comput Methods Biomech Biomed Eng 2014;17(4): 450-458.

[22] Carty G, Chatpun S, Espino DM. Modeling blood flow through intracranial aneurysms: a comparison of Newtonian and non-Newtonian viscosity. J Med Biol Eng 2016;36(3): 396-409.

[23] Karimi A, Navidbakhsh M, Razaghi R, Haghpanahi M. A computational fluid-structure interaction model for plaque vulnerability assessment in atherosclerotic human coronary arteries. J. Appl. Phys 2014;115(14).

[24] Karimi A, Navidbakhsh M, Razaghi R. Plaque and arterial vulnerability investigation in a three-layer atherosclerotic human coronary artery using computational fluid-structure interaction method. J. Appl. Phys 2014;116(6).

[25] Astorino M, Gerbeau JF, Pantz O, Traoré KF. Fluid–structure interaction and multi-body contact: application to aortic valves. Comput Methods Appl Mech Eng 2009;198(45):3603-3612.

[26] Baccani B, Domenichini F, Pedrizzetti G. Vortex dynamics in a model left ventricle during filling. Eur. J. Mech. B/Fluids 2002; 21(5):527-543.

[27] Oncel D, Oncel G, Tastan A. Effectiveness of dual-source CT coronary angiography for the evaluation of coronary artery disease in patients with atrial fibrillation: initial experience. Radiology 2007; 245(3): 703-711.

[28] Tang D, Pedro J, Yang C, Zuo H, Huang X, Rathod RH, Gooty V, Tang A, Wu Z, Billiar KL. Patient-specific MRI-based right ventricle models using different zero-load diastole and systole geometries for better cardiac stress and strain calculations and pulmonary valve replacement surgical outcome predictions. PLoS ONE 2016; 11(9).

[29] Elbaz MS, Van der Geest RJ, Calkoen EE, De Roos A, Lelieveldt BP, Roest AA, Westenberg JJ. Assessment of viscous energy loss and the association with three-dimensional vortex ring formation in left ventricular inflow: In vivo evaluation using four-dimensional flow MRI. Magn Reson Med 2017;77(2): 794-805.

[30] Suito H, Takizawa K, Huynh VQ, Sze D, Ueda T, Tezduyar TE.A geometrical-characteristics study in patient-specific FSI analysis of blood flow in the thoracic aorta. Advances in Computational Fluid-Structure Interaction and Flow Simulation, Springer2016, pp. 379-386.

[31] Lassila T, Malossi C, Stevanella M, Votta E, Redaelli A, Deparis S. Simulation of left ventricle fluid dynamics with mitral regurgitation from magnetic resonance images with fictitious elastic structure regularization. arXiv:1707.03998 [physics.med-ph].

[32] Thomas L, Foster E , Schiller NB. Peak Mitral Inflow Velocity Predicts Mitral Regurgitation Severity. J Am Coll Cardiol 1998;31(1).

[33] Konishi T, Funayama N, Yamamoto T, Hotta D, Kikuchi K, Ohori K, Nishihara H, Tanaka S. Severe mitral regurgitation due to mitral leaflet aneurysm diagnosed by three-dimensional transesophageal echocardiography: a case report. BMC Cardiovasc Disord 2016;16(1): 234.

[34] Vaziri SM, Larson MG, Lauer MS, Benjamin EJ, Levy D. Influence of blood pressure on left atrial size. J Hypertens 1995;25(6): 1155-1160.

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